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Streptococcus sanguis II (Viridans) Prosthetic Valve Endocarditis With Myocardial, Splenic and Cerebral Abscesses

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BACTERIAL ENDOCARDITIS of native and prosthetic heart valves is frequently caused by the viridans group of streptococci. Viridans streptococci are usually sensitive to penicillin, and they are rarely invasive and rarely cause destruction of extracardiac tissue. As a result of these characteristics, bacterial endocarditis caused by this group of bacteria is associated with less mortality and morbidity than endocarditis caused by other organisms.[1-3] *Streptococcus sanguis* is one of the most common species of viridans streptococci causing endocarditis.[4] We present here a fatal case of *S sanguis* prosthetic valve endocarditis that was associated with persistent fever, leukocytosis and myocardial, cerebral and splenic abscesses. This case shows that endocarditis due to *S sanguis* may be associated with an aggressive clinical course.

(Young SG, Davee T, Fierer J, et al: *Streptococcus sanguis II* [viridans] prosthetic valve endocarditis with myocardial, splenic and cerebral abscesses. West J Med 1987 Apr; 146:479-481)

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Report of a Case

The patient, a 64-year-old man, was admitted with a complaint of fatigue, fever and chills of five days' duration. Two years before admission, an Ionescu-Shiley aortic valve prosthesis was implanted because of calcific aortic stenosis and a dual-chamber pacemaker was implanted for perioperative heart block. On admission, the patient was oriented but lethargic, with a temperature of 36°C (96.8°F) and a respiratory rate of 25 per minute. There was one conjunctival hemorrhage, but no Roth spots were seen on funduscopic examination, and no cutaneous signs of endocarditis were observed on the hands or the feet. The jugular venous pressure was normal and on examination of the chest scattered rhonchi were heard. A systolic murmur consistent with the prosthetic aortic valve was noted, but there were no diastolic murmurs or gallops. There was no abdominal tenderness or hepatosplenomegaly.

A urinalysis showed microscopic hematuria. The hematocrit was 36% and leukocyte count was 16,300 per μl with 77% segmented forms, 7% band forms, 14% lymphocytes and 2% monocytes. The serum creatinine level was 4.0 mg per dl. The electrocardiogram showed a paced rhythm, and the chest x-ray film showed mild cardiomegaly without evidence of pulmonary venous congestion. An echocardiogram and Doppler study did not show any valvular vegetations or evidence of valvular regurgitation initially or later during the hospital course. Three of three blood cultures grew viridans streptococci, sensitive to penicillin (minimum bactericidal concentration < 0.1 μg per ml). On subsequent characterization with conventional media and API Rapid Strep (Analytab Products, Inc, Plainview, NY), it was identified as *S sanguis II*.

Antibiotic therapy with penicillin G (6 million units per day) and gentamicin sulfate was begun. Repeat blood cultures were consistently negative. Despite antibiotic therapy, the patient continued to have daily febrile episodes with temperatures of 38.8°C to 39.4°C (102°F to 103°F) throughout the hospital course. The leukocyte count remained between 15,000 and 30,000 per μl. On day 2, his mental status deteriorated to stupor, and on day 5 a right hemiparesis and seizures developed. A computed tomographic (CT) scan of the head revealed bilateral cerebral emboli. There were many petechiae on the feet and hands and new petechial lesions appeared throughout his hospital course. On day 13, biplanar ascending aortography showed a multiloculated perivalvular abscess extending below the prosthetic valve ring (Figure 1), but there was no aortic regurgitation. No left ventriculogram was done. A repeat CT scan of the head on the same day revealed multiple contrast-enhancing cerebral lesions, consistent with new embolic events, and a CT scan of the abdomen showed several large splenic abscesses.

To investigate the possibility that infection with another organism besides *S sanguis* accounted for the aggressive course of the illness, a percutaneous aspiration of one of the splenic abscesses was done. Serosanguineous material containing many polymorphonuclear leukocytes and Gram-positive cocci was obtained, but the culture of this material was sterile, even though penicillinase was added to the agar. Within three hours of the splenic aspiration, a fever to 39°C

(102°F) and hypotension developed and the patient had a pronounced reduction in the calculated systemic vascular resistance. There was no evidence of intraperitoneal bleeding. Respiratory and renal failure ensued, and the patient died on the 21st hospital day. Throughout the hospital course there was continued evidence of recurrent peripheral emboli. At no time during the hospital course did the patient have aortic regurgitation or congestive heart failure.

On postmortem examination there was a large myocardial abscess beneath the prosthetic aortic valve and a sinus tract extending from an abscess cavity in the interventricular septum to the pulmonary artery. Vegetations were noted on the prosthetic aortic valve cusps, the anterior leaflet of the mitral valve and the tricuspid leaflets. The spleen showed several large abscess cavities (Figure 2). The brain showed evidence of multiple thromboembolic lesions with microabscess cavities and masses of bacteria surrounding several arteries. Postmortem cultures of the heart valve and spleen did not yield *S sanguis*.

Discussion

Viridans streptococci are a frequent cause of bacterial endocarditis, both on native valves and prosthetic heart valves, especially on prosthetic valves that have been in place for more than two months.[1-6] In general, patients with these infections respond well to antibiotic therapy and have an excellent prognosis.[1-3,5,6] The main reasons for this excellent prognosis are that viridans streptococci are usually sensitive to penicillin and they rarely invade tissue or form abscesses. Not surprisingly, the cure rate is somewhat lower if a prosthetic valve is infected,[5,6] but even in such cases it is rare for there to be suppurative extracardiac lesions.[3] What distinguished this case of viridans streptococcal endocarditis was the occurrence of multiple splenic abscesses and numerous microscopic brain abscesses. There was also an extensive myocardial abscess, but this complication is known to occur occasionally with prosthetic valve infections, even with relatively avirulent bacteria.[5,7]

It is generally believed that viridans streptococci cannot cause serious extracardiac infections because they are killed by normal host defenses. The relatively avascular heart valve and the fibrin-platelet nidus that precedes infection are areas of the body wherein an inflammatory infiltrate that allows bacteria to proliferate cannot form.[8,9] Despite the release of millions of bacteria from vegetations each day,[10,11] metastatic infection is rare with viridans streptococci and most of the extracardiac damage is due to either emboli or immune-complex vasculitis.[3,9] The only viridans streptococci that are exceptions to this rule are *Streptococcus pneumoniae*[12] and *Streptococcus milleri*.[13] Both of these organisms can be invasive and form abscesses, and endocarditis due to these organisms can be aggressive. *S pneumoniae* organisms are invasive in part because they are protected from phagocytosis by a polysaccharide capsule.[14] *S milleri* has been reported to produce parenchymal abscesses even in the absence of endocarditis.[13,15,16] Their virulence factors are still poorly understood. Although the taxonomic classification of viridans streptococci is still somewhat unsettled, the infecting organism in this case was a typical *S sanguis II* and not *S milleri*.

S sanguis has not been previously reported to have invasive characteristics. Hosea reported three cases of viridans endocarditis associated with abscesses and tissue invasion, but the species of the α-hemolytic streptococci were not reported.[17] Arnett and Roberts[7] and Karchmer and co-workers[5] have reported the occurrence of valve ring abscesses or uncontrolled infection (or both) with viridans streptococci, but neither antibiotic sensitivity nor the exact species of the organisms was reported. The remarkable aspect of our case was that *S sanguis* infection was associated with both myocardial and peripheral abscess formation despite the fact that the organism was exquisitely sensitive to penicillin.

The most common indication for surgical intervention in endocarditis is congestive heart failure, usually a result of acute valvular regurgitation.[18-20] Infection of a prosthetic valve with a virulent or aggressive organism or an organism that is relatively resistant to antibiotics is an indication for surgical intervention.[1,5,6] Surgical intervention in this case was delayed because the patient had no valvular regurgitation or congestive heart failure and because we had identified the infecting organism as *S sanguis II* and knew that it was sensi-

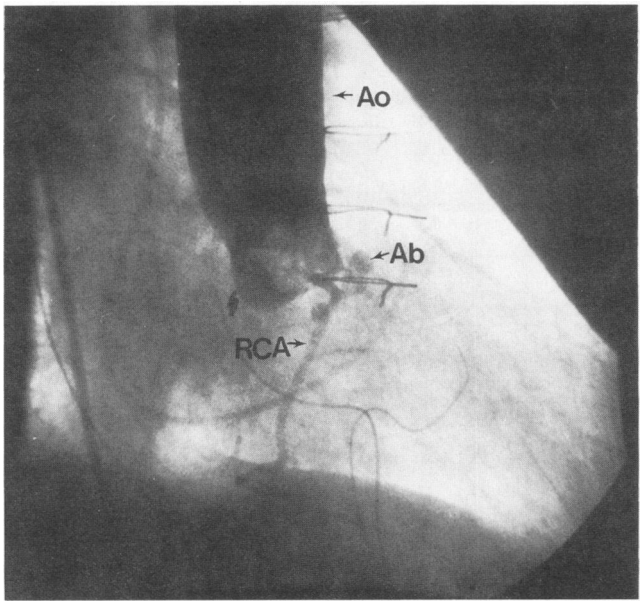

Figure 1.—Right anterior oblique ascending aortogram. Injection of contrast material into the ascending aorta showed a normal ascending aorta **(Ao)** and no evidence of aortic valve regurgitation. A small multiloculated abscess cavity **(Ab)** could be seen beneath the prosthetic aortic valve. Further angiographic studies showed that the abscess did not involve the origin of the right coronary artery **(RCA)**.

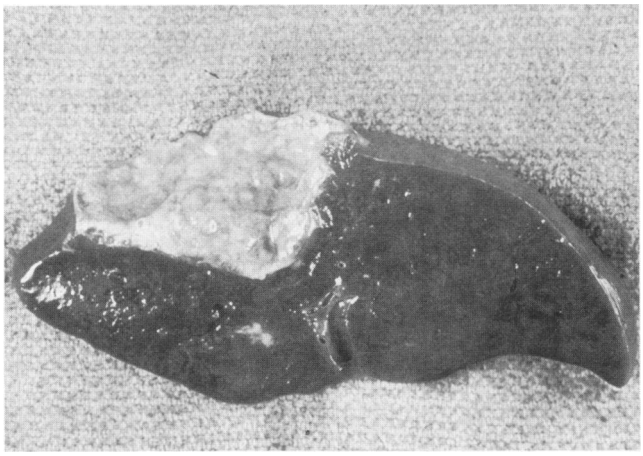

Figure 2.—The photograph shows the spleen at necropsy. A large abscess cavity is seen in this section of the spleen. Other abscess cavities were seen in other sections of the spleen.

tive to penicillin. The persistent fever, however, suggested uncontrolled infection, even though all blood cultures after therapy were negative. Karchmer and colleagues found that fever lasting more than nine days after initiation of therapy was associated with a high incidence of myocardial abscess and a poor prognosis, as it was in this case.[5] Unfortunately, our patient sustained severe neurologic damage from multiple embolic events before surgical treatment could be undertaken. Had the cardiac abscess been identified earlier, a cardiac operation might have prevented the cerebral emboli.

This patient had numerous splenic abscesses on the computed tomographic scan of the abdomen. Johnson and coworkers have suggested that computed tomography is the best radiographic modality for detecting splenic abscesses.[21] Splenic abscesses occur occasionally in patients with infective endocarditis, but they are extremely rare with viridans streptococci.[22] In general, treatment of splenic abscesses associated with endocarditis requires splenectomy and antibiotic therapy; antibiotic therapy alone is not sufficient.[22] Whether our patient would have recovered from the splenic abscesses without an abdominal operation is uncertain. It is likely, however, that the abscesses in this patient would have been cured by antibiotic therapy alone because they were sterile when aspirated on day 13 of treatment and bacteria could not be cultured from the spleen at the postmortem examination.

Conclusions

S sanguis II, a viridans streptococcus, may be associated with tissue invasion and abscess formation in a case of prosthetic valve endocarditis. Persistent fever and leukocytosis with multiple embolic events should prompt consideration of surgical intervention, even in the absence of positive blood cultures, valve dysfunction or congestive heart failure.

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Sarcoidosis Presenting as Chronic Thrombocytopenia

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THROMBOCYTOPENIA is a rare complication of sarcoidosis. It may be immune in origin,[1] but increased platelet-associated immunoglobulin (Ig) G levels has been reported in only three previous cases.[2-4] We present a case of a patient with sarcoidosis and chronic thrombocytopenia with increased platelet-associated IgG levels.

Report of a Case

The patient, a 23-year-old man, was admitted to hospital in August 1983 for investigation of thrombocytopenia. The patient first came to medical attention when a large thigh hematoma developed in 1977. His platelet count was 63 × 10^9 per liter at that time. The hemoglobin and leukocyte counts were normal. No other laboratory tests were done.

A knee operation in 1979 was uncomplicated, at which time his hemoglobin value and leukocyte count were normal. Platelets were not counted.

This admission was for drainage of a supraorbital hematoma. The patient said he did not have easy bruising or abnormal bleeding aside from these two episodes. He also said he did not have other health problems and was not taking medications. Apart from the hematoma, a physical examination showed no abnormalities.

The platelet count was 16 × 10^9 per liter. The hemoglobin level was 2.53 mmol per liter (16.3 grams per dl), the leukocyte count was 6.5 × 10^9 per liter with a normal differential, and giant platelets were noted on the blood smear. A bone marrow aspirate and a biopsy specimen showed megakaryocytic hyperplasia. The value of platelet-associated IgG, assayed by a radial immunodiffusion technique,[4] was 9.9 fg per platelet (normal 3.1 ± 2.6 fg per platelet; mean ± standard deviation). The prothrombin time and partial thromboplastin time were normal. A chest roentgenogram showed bilateral hilar adenopathy. Pulmonary function and serum angiotensin-converting enzyme and serum calcium levels were normal.

(Field SK, Poon MC: Sarcoidosis presenting as chronic thrombocytopenia. West J Med 1987 Apr; 146:481-482)

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